THE EFFECT OF TEMPERATURE ON THE SKIN[1]

A Review

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The temperature of the skin may be varied readily according to the environment to which it is exposed. Even if the whole surface be exposed to a constant environment the skin temperature varies widely in different areas. The great variations commonly found in different areas depend mainly on adjustments of the circulation which regulate the degree of heat loss from the limbs. The limbs have relatively large surfaces and the skin temperature may be modified not only by variations in the amount of blood supplied, but also by variations in the paths by which the blood is conducted.

If the individual is in thermal equilibrium, the average temperature of the surface is determined by the amount of heat transferred and the effective thermal conductivities on either side of the surface. The surface forms the boundary between the body and the environment, and the heat it acquires from the body must equal that lost to the environment. The former depends on the internal thermal gradient and the effective thermal conductivity of the tissues, the latter on the external thermal gradient and the external effective thermal conductivity (taking account of all modes of heat loss whether by conduction or otherwise) (1).

In *a cold environment* this external thermal gradient is considerable and the thermal conductivity is not under control. Control can therefore only be attained by lowering the average

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external gradient by reducing the skin temperature. This reduction in external gradient is attained most readily in the peripheral parts of the limbs. Through vasoconstriction the blood-flow is reduced, and the blood cooled at the surface is returned mainly through the venae comites, for the superficial veins are constricted. The blood passing peripherally through the arteries is subjected to the cooling effect of the blood returning in the venae comites; thus a low surface temperature is attained with a circulation to the skin adequate for nutrition. Though the blood is cooled before it reaches the surface much of the heat is transferred to returning venous blood and is not lost from the body. The cooling effect is also extended by the same means to the tissues lying beneath the skin, and the shape of the internal gradient is altered. The total changes achieved are a reduction in the external gradient between the skin and the environment by lowering of the surface temperature; an increased rectal to skin thermal gradient which is attained through the development of a low thermal conductivity by vasoconstriction and lowered blood flow as well as by the direction utilized for the return of blood; an alteration in the shape of the internal thermal gradient such that it is spread over a greater depth, and is consequently less steep in the most superficial areas (2), a change also attained by the direction utilized for blood flow. By all these changes heat is conserved. The effect produced by altering the direction of blood flow is not a new discovery; it was recognized by Claude Bernard in 1876 (3).

If an area be exposed to extreme cold (e.g. immersion of a limb in stirred water at or below 15°C.) a hyperemia of bright red color may be seen. This involves the smaller skin vessels, is probably paralytic in origin, and is not associated with increased flow (4). The red color depends on a metabolic activity so lowered by cold as to leave the blood oxygenated. Increased flow may however occur in the most peripheral areas, such as the fingers, and result in temporary rises in skin temperature (5). This probably depends on an intermittent dilatation of arterio-venous anastomoses which in emergencies can protect peripheral areas from damage from over-cooling, though at the

expense of general bodily economy (6). Lefevre (7) earlier had noted the hyperemic response to cold and associated it with an increased blood-flow and increased heat loss. He probably arrived at erroneous conclusions for two reasons: (a) any increase in blood flow is limited to small areas (b) increased heat loss can occur without increased flow since the skin is supplied by vessels which penetrate the muscles. If the muscles are active, whether voluntarily, or involuntarily as in shivering, their increased heat causes the blood supplying the skin to be warmer. This mechanism, useful no doubt in exercise, is inefficient during shivering (8).

The *vascular reactions to warmth* are the opposite of those to cold, except that the arterio-venous anastomoses are also opened to warmth, and may then remain open more or less continuously (6) (9). According to the Clarks (9) the number of such anastomoses in the rabbit's ear does not appear to be fixed but actually increases during prolonged exposure of the animal to warmth. The total flow of blood to the skin may be increased to the order of about 10 times that obtaining at low temperatures (10). In addition venous return is largely maintained through superficial venous channels, where the returning blood is still further cooled. The arterial blood in its path down the arm is less exposed to cooled blood, so that a high skin temperature is more readily attained, and the thermal gradient in the dermis is steepened. The total changes achieved in such adjustments are an external thermal gradient between the skin and the environment increased through a raised surface temperature; an increased external effective thermal conductivity obtained by greater heat loss through evaporation; a decreased rectal to skin thermal gradient which is attained through an increase in the effective thermal conductivity of the tissues; an alteration in the shape of the internal gradient so that it is steepest in the most superficial layers, and warm blood is brought close to the surface (2); additional cooling of blood during its return passage along superficial veins.

Through these mechanisms the surface temperature of the hands and fingers, and consequently the heat loss, can be regu-

lated through a wide range. Under conditions of exposure to cold the temperatures of these surfaces may be much below those of the face and trunk. Under conditions when heat loss is difficult, as for instance in a normal subject exposed to a warm environment, or in a hyperthyroid patient in a normal environment, the surface temperatures of the hands and fingers may be quite high, exceeding those of the face and trunk (11 and 12). Values may then be found that exceed 36°C. (97°F.).

Vasodilatation to warmth in any area raises the *capillary pressure* (13) and must therefore raise the rate of *fluid transudation.* In consequence the rate of edema formation is increased, if the conditions are favorable for its occurrence. This is certainly the case (14) when venous pressure is raised either by venous obstruction or through dependency of the limb without muscular movements. It is probably also the case when the permeability of the vessels is altered through inflammation. Heat is therefore apt to cause not only more rapid blood flow but also more rapid swelling of injured or inflamed areas. Fluid transuded from the skin vessels also tends to keep the epidermis more moist. It has been shown by Gagge (15) that the degree of wetness of the skin varies and that these variations are utilized to regulate heat loss through evaporation. There is a close parallelism between the degree of wetness of the skin and the skin circulation as measured by its effective thermal conductivity (16). This relationship appears to hold in maximal vasoconstriction, in moderate vasodilatation where sweating is unlikely to be present, as well as at higher temperatures where sweating is undoubtedly a factor.

Moistness of the skin is probably dependent, at lower temperature levels *where sweating is not present,* almost entirely on fluid transudates from the vessels. Its variation with circulatory conditions is consequently understandable. The actual quantity of water loss is probably regulated by the amount of fluid filtered and is little affected by the humidity of the environment. The moistness of the skin is on the contrary much affected by the environment; if the air is dry the epidermis is relatively dry and protects the deeper layers of the skin; if the air is moist a

greater wetness of the epidermis enables fluid to be evaporated in almost normal amount in spite of a greater humidity of the environment. Thus at a temperature of 31°C. (87.8°F.) with dry air the wetness of the skin averages 10 per cent, that is to say the evaporation is 10 per cent of that which would occur from a completely wet surface; in air with 65 per cent humidity at the same temperature the wetness of the skin is twice as great (17). Such values represent averages; the wetness of the skin varies in different areas. Where two surfaces are in contact, as between the fingers or toes or in folds of the buttocks, there may be little or no evaporation and all the layers of the skin may be more or less moist. Contact of two such surfaces also raises their temperature, but only slightly, for the entering blood has already been cooled to some extent in the deeper layers of the skin and subcutaneous tissue.

Under conditions of warmth, *where sweating occurs*, the moistness of the skin is obviously determined mainly by the sweat secreted and by the conditions for its evaporation. Such secretions may be distinguished from evaporation of fluid transudates since secreted sweat contains chloride which is carried to the surface with the fluid. The concentration of this chloride is approximately 0.2 per cent NaCl. This concentration is said to diminish as a subject is exposed to continued heat under conditions when chloride retention should be advantageous (18). The data however only demonstrate that the ratio of water loss to salt loss from the skin is increased. Such a change could depend merely on an increased loss of water from fluid transudates.[3]

Water loss from the hands and feet appears to be different from that from other areas of the body. It is more continuous and is less dependent on thermal stimulation, but may be increased with emotion. The moist sweaty palms produced by excitement are familiar objects. According to Kuno (19) the secretion of sweat from the hands and feet is continuous and serves to keep sensory surfaces adequately moist and sensitive; possibly this secretion is controlled by adrenergic sympathetic

[3] More direct evidence is submitted in a later paper. Dill, D. B., Hall, F. G. and Edwards, H. T. Amer. Jour. Physiol., **123**: 412, 1938.

fibers, while certainly the sweat glands as a whole are controlled by cholinergic sympathetic fibers. Whether due to sweat secretion, evaporation of fluid transudates, or to both factors, the water evaporated from the skin of the hands and feet represents a large proportion of the total insensible loss from the skin. According to Benedict and Wardlaw (20) it accounts for 30 per cent of the total basal skin loss, and the rate of water loss from the skin of the hands and feet is approximately 3 times as great per unit area as compared with that from the rest of the body.

The *metabolic activity* of the skin varies with its temperature, but in any case it is always low relative to the circulating blood. The minimal oxygen saturation of blood leaving the skin is found when the skin temperature is about 25°C. (79°F.) as was pointed out by Goldschmidt and Light (4); but even so venous blood coming entirely from the skin tissues of the hand does not have an oxygen saturation below 71 per cent and rarely one below 82 per cent (4 and 21). Similarly, in dogs, the blood returning from the paw shows an oxygen saturation which is minimal at skin temperature of about 20°C. but even so does not fall below 75 per cent (22). The minimal saturation is seen at temperatures low enough to depress the circulation considerably, but not low enough to cause as great a depression of skin metabolism (4).

The effect of temperature changes on the *physico-chemical conditions in the blood* is very great. If the blood be cooled, the effect on the dissociation constants of proteins acting as acids is much greater than that on the dissociation constant of carbonic acid. In consequence if blood be cooled at constant CO_2 content, base is displaced from combination with protein to combine with carbonic acid, and bicarbonate is thus increased, while free carbonic acid (and tension of CO_2) is decreased. Consequently the pH shifts considerably to the alkaline side. At the same time the solubility of CO_2 in plasma is increased, and this assists in a minor way to lower the tension of CO_2. With the reduction in temperature the dissociation of water into H^+ and OH^- ions is also less, so that the hydrogen ion concentration for neutrality is reduced; the pH value for neutrality is increased. Increase

in pH and lowering of the temperature both decrease the tendency of oxy-hemoglobin to unload oxygen, consequently oxygen tension is reduced. The opposite effects are seen on heating. These physical effects were first discussed in detail by Austin and Cullen (23); they have been demonstrated to be effective in vivo (21 and 24) and further analysis of the physico-chemical changes involved may be found in later papers (25 and 26).

The temperature changes affect the *gas tensions of oxygen and carbon dioxide* as the result both of the alteration in the ratio of blood flow to metabolism and the consequent effect on the venosity of venous blood, and also of the changes in the physico-chemical constants of blood. The combined effect of these two factors has been investigated in the subcutaneous tissue of the hand and forearm by the subcutaneous injection of gas mixtures. Such mixtures after an hour or so come into equilibrium with the blood in the subcutaneous tissues, and if removed and analyzed consequently indicate the gas tensions in this tissue (24). Such gas tensions are found to be in agreement with those calculated from blood drawn from cutaneous veins (21). The tension of carbon dioxide so determined ranges from 20 mm. Hg. at a skin temperature of 10° to 14°C. (50–57°F.) to 47 mm. at a skin temperature of 41° to 44°C. (106–111°F.). It varies lineally with skin temperature. The tension of oxygen ranges in a complicated curve from 18 to 23 mm. at a skin temperature of 10° (50°F.) to a minimum of 15 to 25 mm. at a skin temperature of 22° (72°F.) and then rises to a maximum of 87 mm. Hg. at a skin temperature of about 42° (108°F.). The changes in the subcutaneous tissue are therefore very great; those in the skin itself should be even greater. The fact that in a cooled area the carbon dioxide tension of the tissues and of venous blood can be much below that in arterial blood in the lungs is at first glance surprising. However it may be readily understood when one considers that the arterial blood in the area concerned is also cooled and has a still lower CO_2 tension, while the venous blood removed from the vein has only to be warmed to show a normal high CO_2 tension.

The values for the gas tensions of CO_2 and O_2 that have been

cited are certainly somewhat abnormal due to the inflammatory reaction to the injection of air. Real CO_2 values are probably slightly higher and O_2 values lower. However inflammatory reactions are slight when a limb is cooled and they are of no great significance in the presence of the marked hyperemia produced by heat. The marked variations produced by temperature changes are real. The actual level of the gas tensions at ordinary skin temperatures are less certain. Values for the subcutaneous tissue of the abdomen, where the skin temperature is not very variable, have been obtained by Seevers (27) by a chronic method. At a skin temperature of 35°–36° the values for O_2 and CO_2 tension in acute experiments in the forearm are about 62 and 38 mm. respectively; in the abdomen the chronic experiments of Seevers show that they are 22 mm. and 45 mm. In chronic experiments the circulation may be impaired by cicatrization; the real normal values probably lie between these two sets of figures.

Since the gas tensions of oxygen may be modified by a factor of 4 times according to the temperature, it is probable that the temperature of the tissue must modify the capacity of anaerobic organisms to develop and with such infections it would seem logical to maintain the local temperature as high as possible provided that the circulation is not obstructed. Whether the pH changes (representing increases of 0.4 or more) that are attainable by cold are of importance in infection is less certain. The change in pH of neutrality is in the same direction and of approximately the same order, so that the difference between the hydrogen ion concentration of the blood and that of a neutral solution is not much altered.

The *mechanisms by which the vascular reactions* to heat *are controlled* are probably multiple. The vessels are to some extent regulated by chemical factors, for dilatation to warmth and constriction to cold may be seen in sympathectomized or even in completely denervated areas (28 and 29). There is some evidence that blood is supplied in accordance with the oxygen consumption, though the evidence is not conclusive (22). Some metabolic product may be involved, or the physico-chemical changes in blood that accompany a change in temperature may

be responsible. In addition to such local factors a general dilatation of the skin vessels may be induced through nervous channels as the result of a rise in temperature in the central nervous system, probably in the hypothalamic region. When a limb is warmed, such a general dilatation only develops after some delay and after the rectal temperature has begun to rise; consequently the mechanisms involved are considered to be central. This response necessitates the presence of sympathetic nerves; probably there is inhibition of vasoconstrictor tone and active dilatation through sympathetic vasodilator nerves (30). In addition, dilatation may probably be produced through reflex mechanisms, for relatively slight increases in skin temperature (of 0.5°C. or less) may induce vasodilatation. Such reactions cannot depend on a rise in brain temperature, for their development is accompanied by a fall of rectal temperature (8). It is unlikely that such small changes in temperature affect significantly the level of local metabolism. Consequently the mechanism involved must be assumed to be reflex. The motor mechanisms involved are presumably the same as those concerned in response to a central increase in temperature. At higher temperature levels both of the latter methods of response may be associated with sweating. Drinking of water at body temperature is said to cause a considerable increase in water loss from the skin through reflex mechanisms; this is interpreted as an increased sweat secretion (31).

SUMMARY

The temperature of the skin is varied according to the external environment to which it is exposed, and the vascular adjustments in the subjacent areas. The latter adjustments consist of variations in arteriolar, capillary and venous tone, with alterations both in the quantity of blood flowing to the skin, and also in the return path mainly utilized. This path may, or may not, allow cooling of the arterial blood before it reaches the surface. In the extremities variations in the utilization of arterio-venous anastomoses also occur. With extreme cold, dilatation of capillaries with arteriolar constriction and slow blood-flow may develop.

Vascular dilatation is associated with a rise in capillary pres-

sure, increased fluid transudation, and an increased wetness of the skin, even in the absence of sweat secretion. If edema is developing as a result of stasis or inflammation, a rise of temperature probably increases the rate at which fluid collects. Per unit of time the water transuded into the skin and evaporated may be independent of the humidity of the environment, within certain limits. But this is not true of the moistness of the skin. It is by variations in this moistness that the water-loss may be kept constant over a wide range of conditions. Water-loss from the skin of the hands and feet is three times as great per unit of surface area as that in the rest of the body. There is some evidence that the sweat from the hands and feet differs also from that of the rest of the body, in that it appears to be particularly affected by emotional states.

Changes in temperature in an area involve considerable alterations in the physico-chemical state of the blood, as well as in its rate of circulation. Cooling increases the pH and this increase may exceed 0.4 units; the pH value of neutrality is also increased. As the result of a decreased binding capacity for base on the part of protein, base bicarbonate is increased and free carbonic acid is reduced. This change with an increased solubility for CO_2 reduces CO_2 tension. The resultant effects of changes in pH and temperature reduce the O_2 tension at low temperatures and oxygen tension may vary 300 to 400 per cent.

The reactions of vasodilatation and sweating can probably be induced both as the result of a rise in temperature in the hypothalamus, and as a reflex response to a wide-spread stimulation of receptors for warmth. Vasomotor reactions may develop locally as the result of chemical changes in the blood or in the tissues, acting directly on the vessels.

REFERENCES

(1) Burton, A. C.: The application of the theory of heat flow to the study of energy metabolism. Jour. Nutrit., 7: 497–533, 1934.

(2) Bazett, H. C. and McGlone, B.: Temperature gradients in the tissues in man. Amer. Jour. Physiol., 82: 415–451, 1927.

(3) Bernard, Claude: Leçons sur la Chaleur animale, Paris, 1876.

(4) Goldschmidt, S. and Light, A. B.: A comparison of the gaseous content of blood from veins of the forearm and the dorsal surface of the hand

as indicative of blood flow and metabolic differences in these parts. Amer. Jour. Physiol., **73**: 127–145, 1925.

GOLDSCHMIDT, S. AND LIGHT, A. B.: The effect of local temperature upon the periperal circulation and metabolism of the tissues as revealed by the gaseous content of the blood. Amer. Jour. Physiol., **73**: 146–172, 1925.

(5) LEWIS, T.: Observations upon the reactions of the vessels of the human skin to cold. Heart, **15**: 177–208, 1930.

(6) GRANT, R. T. AND BLAND, E. F.: Observation on arteriovenous anastomoses in human skin and in the bird's foot with special reference to the reaction to cold. Heart, **15**: 385–411, 1931.

GRANT, R. T., BLAND, E. F. AND CAMP, P. D.: Observations on the vessels and nerves of the rabbit's ear with special reference to the reaction to cold. Heart, **16**: 70–103, 1932.

(7) LEFÈVRE, J.: Chaleur Animale, Paris, 1911.

(8) BURTON, A. C. AND BAZETT, H. C.: A study of the average temperature of the tissues, of the exchanges of heat, and vasomotor responses in man by means of a bath calorimeter. Amer. Jour. Physiol., **117**: 36–54, 1936.

(9) CLARK, E. R. AND CLARK, E. L.: The new formation of arteriovenous anastomoses in the rabbit's ear. Amer. Jour. Anat., **55**: 407–467, 1934.

(10) PRINZMETAL, M. AND WILSON, C.: The nature of the peripheral resistance in arterial hypertension with special reference to the vascular system. Jour. Clin. Invest., **15**: 63–83, 1936.

(11) BENEDICT, F. G.: Die Temperatur der menschlichen Haut Ergeb. d. Physiol., **24**: 594–617, 1925.

(12) BROWN, C. L.: Trans. College of Physicians, Philadelphia, 1938.

(13) LANDIS, E. M.: Capillary pressure and capillary permeability. Physiol. Rev., **14**: 404–481, 1934.

(14) LANDIS, E. M. AND GIBBON, J. H., JR.: The effects of temperature and of tissue pressure on the movement of fluid through the human capillary wall. Jour. Clin. Invest., **12**: 105–138, 1933.

(15) GAGGE, A. P.: A new physiological variable associated with sensible and insensible perspiration. Amer. Jour. Physiol., **120**: 277–287, 1937.

(16) WINSLOW, C. E. A., HERRINGTON, L. P. AND GAGGE, A. P.: Physiological reactions of the human body to various atmospheric humidities. Amer. Jour. Physiol., **120**: 288–299, 1937.

(17) GAGGE, A. P., HERRINGTON, L. P. AND WINSLOW, C. E. A.: Thermal interchanges between the human body and its atmospheric environment. Amer. Jour. Hyg., **28**: 84–102, 1937.

(18) DALY, C. AND DILL, D. B.: Salt economy in humid heat. Amer. Jour. Physiol., **118**: 285–289, 1937.

(19) KUNO, Y.: The significance of sweating in man. Lancet, 1930 (1), 912–915.

(20) BENEDICT, F. G. AND WARDLAW, H. S. H.: Some factors determining the insensible perspiration of man. Arch. Int. Med., **49**: 1019–1031, 1932.

(21) SRIBYATTA, L. AND BAZETT, H. C.: The effect of local changes in temperature on blood from the hand veins. Amer. Jour. Physiol., **86**: 578–586, 1928.

(22) FREEMAN, N. F. AND ZELLER, J. W.: The effect of temperature on the volume flow of blood through the sympathectomized paw of the dog with

observations on the oxygen content and capacity, carbon dioxide content and pH of the arterial and venous blood. Amer. Jour. Physiol., **120**: 475–485, 1937.

(23) Austin, J. H. and Cullen, G. E.: Hydrogen ion concentration in health and disease, Williams & Wilkins, Baltimore, 1926.

(24) Bazett, H. C. and Sribyatta, L.: The effect of local changes in temperature on gas tensions in the tissues. Amer. Jour. Physiol., **86**: 565–577, 1928.

(25) Adair, G. S., Cordero, N. and Shen, T. C.: The effect of temperature on the equilibrium of CO_2 in blood and the heat of ionization of hemoglobin. Jour. Physiol., **67**: 288–298, 1929.

(26) Roughton, F. J. W., Adair, G. S., Barcroft, J., Goldschmidt, S., Herkel, W., Hill, R. M., Keys, A. B. and Ray, G. B.: The thermochemistry of the oxygen-haemoglobin reaction. Biochem. Jour., **30**: 2117–2133, 1936.

(27) Seevers, M. H.: O_2 and CO_2 tension in the subcutaneous tissues of normal subjects. Amer. Jour. Physiol., **115**: 38–42, 1936.

(28) Freeman, N. E.: The effect of temperature on the rate of blood flow in the normal and sympathectomized hand. Amer. Jour. Physiol., **113**: 384–398, 1935.

(29) Trotter, W. and Davies, H. M.: Experimental studies in the innervation of the skin. Jour. Physiol., **38**: 134–246, 1909.

(30) Lewis, T. and Pickering, G. W.: Vasodilation in the limbs in response to warming the body; with evidence for sympathetic vasodilator nerves in man. Heart, **16**: 33–52, 1931.

Pickering, G. W.: The vasomotor regulation of heat loss from the skin in relation to external temperature. Heart, **16**: 115–135, 1932.

(31) Lee, D. H. K. and Mulder, A. G.: Some immediate effects of reduced cooling powers upon the water balance and related effects in the human subject. Jour. Physiol., **84**: 410–432, 1935.

DISCUSSION

Dr. Theodore Cornbleet, *Chicago:* I wish I lived more closely to Dr. Bazett because he is an expert in some things along which I need help. I can corroborate what he says about the chloride not being constant. I have been doing some experiments in which serial specimens are examined during one sweating bout, and found the chloride concentration of the sweat by no means constant. The chloride tends to increase for a definite period of time until a maximum is reached and then it tends to decrease rather markedly. A number of years ago when I was an assistant to Dr. Engman in St. Louis where it gets quite warm, we were impressed by the numbers of impetigo cases we had in the month of August. To that end we set ourselves to analyzing statistically our cases and found that the numbers of impetigo cases did not vary so much with the temperature but did very definitely with the humidity. The humidity, while not entirely to blame, was a major factor. Another point I wish to touch upon is the matter of heat and sugar in the skin. Some of us are constantly harking back to the sugar problem. We refuse to be downed in spite of the fact that many observers find that the sugar in the blood is not increased in pyodermic conditions. On the other hand I find that cutaneous sugar does make a difference in regard to pyodermic infec-

tions in spite of the fact that the blood sugar may not be raised. I find there is a rather marked increase in the sugar content of the skin in warm weather. This can be easily demonstrated in a heat chamber. Looking at Dr. Bazett's figures I am just wondering whether the increased acidity present when the skin is heated may be a factor in hastening glycogenolysis. I have data which show how heat hastens sugar formation from glycogen in the skin and this increased acidity that Dr. Bazett shows to follow heat would tend to explain my results.

DR. J. GARDNER HOPKINS, *New York City:* I cannot discuss this paper but would like to ask Dr. Bazett for a little more information as to the increased sweating of the hands and feet. A patient who is getting fever therapy turns on his sweat all over the body with almost mathematical accuracy when the rectal temperature goes up to a certain point. I always supposed sweating of the hands and feet occurs at the same time. Is this the case?

DR. H. C. BAZETT, *Philadelphia:* The ratio of chloride to water loss from the skin is certainly variable. Since some water may be lost from the skin by fluid transudates from the vessels, it is unsafe at present to state that the chloride concentration of the secretion of the sweat glands varies. With local heating the blood becomes more acid because protein then has an increased capacity to bind base and so decreases plasma bicarbonate and increases carbonic acid. When the whole body is heated, some hyperpnoea results, large amounts of CO_2 are eliminated, and the blood becomes more alkaline.

Sweat glands are certainly mainly innervated by cholinergic nerves distributed by the sympathetic system. Probably some of the glands, particularly some of those in the hands, may also be controlled by adrenergic sympathetic nerves. Whether the same glands can be innervated by both types of nerve is uncertain.

For measurement of skin temperature Hardy's radiation method is ideal, but Hardy's criticisms of thermocouple methods are only warranted in the case of couples made of heavy wire such as those he and Benedict used. If very light wire couples are used the error becomes very small. We have commonly employed thermocouples of manganin and constantan wire of 6 to 8 1/100 cm. diameter to obtain a low thermal conductivity. Hardy tested and criticized couples of copper and constantan wire of 0.51 mm. diameter which have a thermal conductivity 2,600 times as great. The problem is mainly one of convenience and the accuracy desired. Heavy thermocouples are convenient but inaccurate; light thermocouples are accurate but fragile; Hardy's radiation method can be very accurate but the apparatus is less simple.